bile duct cancers, has not been univocally demonstrated (Gamble 1994).

The putative increased risk of bile duct cancer in subjects exposed to asbestos may be due to different mechanisms. The asbestos fibers cross the alveolar barrier by inhalation or penetrate the gastrointestinal mucosa by ingestion. They then reach the interstitial environment and circulatory system through lymphatic vessels and are finally delivered to all tissues, namely the liver and bile ducts (Miserocchi et al. 2008), where they may start a malignant transformation process (Wingren 2004). In addition, asbestos fibers may reach the bile ducts through the papilla of Vater from the intestinal lumen by retrograde reflux, as do bacteria, and remain in the gallbladder for a long time.

In the near future we may have to consider asbestos as another factor accounting for the etiopathogenesis of cholangiocarcinomas that may explain the otherwise mysterious increasing incidence of intrahepatic cholangiocarcinomas in Western countries.

The authors declare they have no competing financial interests.

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Errata

In the letter by O'Brien et al. [Environ Health Perspect 117:A385–386 (2009)], the competing financial interest declaration was incorrect. The correct declaration is as follows:

> Karen Peabody O'Brien is executive director of Advancing Green Chemistry, a not-for-profit organization that receives support from several private foundations (listed online at http://www.AdvancingGreenChemistry.org/AdvancingGreenChemistry/About_Us.html) to support efforts to build the field of green chemistry. J.P. Myers is founder, chief executive officer, and chief scientist for Environmental Health Sciences (EHS), a not-for-profit organization that receives support from several private foundations (listed online at http://www.environmentalhealthnews.org/about.html) to support EHS's mission to advance public understanding of environmental health sciences. John Warner is president of Warner Babcock Institute for Green Chemistry, a private company that applies the principles of green chemistry in the synthesis of new materials and the redesign of chemical processes.

In the letter by Wilson and Schwarzman [Environ Health Perspect 117:A386 (2009)], the last sentence in the first paragraph was incorrect. The corrected sentence is as follows:

> We would add that public policy that accurately reflects current science—and the needs of the chemicals market—is instrumental to the widespread adoption of green chemistry.

EHP apologizes for the error.

In the article by La Merrill et al. [Environ Health Perspect 117:1414–1419 (2009)], the keys in Figure 3B and Figure 5C should have been in Figure 3C and Figure 5D, respectively. The corrected figures are provided below.

EHP apologizes for the errors.

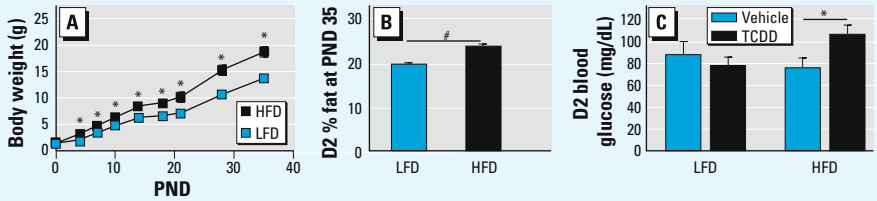

Figure 3. Diet and maternal TCDD exposure effects on body composition and fasting blood glucose. (*A*) HFD increased postnatal D2 body weights (mean ± SE; n = 27–31 at PNDs 0–26 for HFD, and n = 28 at PND35 for LFD). (*B*) HFD (n = 26 mice) increased percent fat at PND35 relative to LFD (mean ± SE; n = 28 mice). (*C*) Fasting blood glucose was increased by HFD and maternal TCDD-treated (n = 5 litters) compared with HFD and maternal vehicle-treated (n = 6 litters) female progeny at PND36 (mean ± SE). Because diet, but not TCDD, changed body weight and percent body fat, these analyses were done on individual D2 mice, with TCDD- and vehicle-treated D2 mice pooled within diet.
*$p < 0.05$. #$p < 0.0001$.

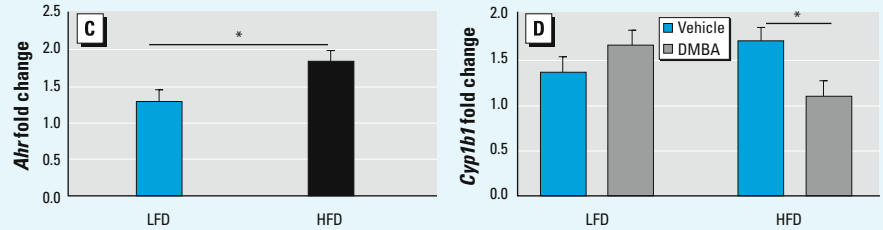

Figure 5. Maternal TCDD exposure and effect of diet on gene expression. Normalized message levels are represented as mean ± SE. (*C*) Induction of *Ahr* was increased by HFD relative to LFD (n = 11 and 10 litters, respectively). Measurements were pooled across TCDD and DMBA groups. (*D*) Induction of *Cyp1b1* by DMBA was decreased compared with vehicle in HFD-fed but not in LFD-fed D2 mice. LFD groups are vehicle (n = 5 litters) and DMBA (n = 5 litters); HFD groups are vehicle (n = 6 litters) and DMBA (n = 5 litters).
*$p < 0.05$.